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Maternal investment, life histories, and the evolution of brain structure in primates

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Maternal investment, life histories, and the evolution of brain structure in primates

Lauren E Powell^{1*}, Robert A Barton¹ & Sally E Street^{1*}

¹ Evolutionary Anthropology Research Group, Department of Anthropology, University of Durham, South Road, Durham, DH1 3LE, UK

*Corresponding authors

Contact details:

Lauren E Powell: powell.laurenElizabeth@gmail.com

Lauren Powell ORCID: 0000-0002-2041-5436

Sally E Street: sally.e.street@durham.ac.uk

Sally Street ORCID: 0000-0001-8939-8016

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Maternal investment, life histories, and the evolution of brain structure in primates

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Abstract

Life history is a robust correlate of relative brain size: larger-brained mammals and birds have slower life histories and longer lifespans than smaller-brained species. The Cognitive Buffer Hypothesis (CBH) proposes an adaptive explanation for this relationship: large brains may permit greater behavioural flexibility and thereby buffer the animal from unpredictable environmental challenges, allowing for reduced mortality and increased lifespan. In contrast, the Developmental Costs Hypothesis (DCH) suggests that life-history correlates of brain size reflect the extension of maturational processes needed to accommodate the evolution of large brains, predicting correlations with pre-adult life history phases. Here we test novel predictions of the hypotheses in primates applied to the neocortex and cerebellum, two major brain structures with distinct developmental trajectories. While neocortical growth is allocated primarily to pre-natal development, the cerebellum exhibits relatively substantial post-natal growth. Consistent with the DCH, neocortical expansion is related primarily to extended gestation while cerebellar expansion to extended post-natal development, particularly the juvenile period. Contrary to the CBH, adult lifespan explains relatively little variance in whole brain or neocortex volume once pre-adult life history phases are accounted for. Only the cerebellum shows a relationship with lifespan after accounting for developmental periods. Our results substantiate and elaborate on the role of maternal investment and offspring development in brain evolution, suggest that brain components can evolve partly independently through modifications of distinct developmental phases, and imply that environmental input during post-natal maturation may be particularly crucial for the development of cerebellar function. They also suggest that relatively extended maturation times provide a developmental mechanism for the marked expansion of the cerebellum in the apes.

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Introduction

Extended lifespan is one of the most consistent correlates of large brain size across mammal species (e.g. (1, 2)). Despite being first identified over 100 years ago (1) and confirmed by multiple comparative analyses since (e.g. (2, 3)), the biological significance of the brain size-lifespan correlation remains uncertain. The Cognitive Buffer Hypothesis (hereafter “CBH”), posits that larger brains bestow behavioural flexibility, which in turn reduces mortality by enabling individuals to adjust to environmental contingency and

unpredictability, and that this reduction in mortality facilitates longer lifespans (1, 2, 4, 5). A variant of the CBH proposes an alternative causal scenario in which longer lifespans create conditions favouring the evolution of enlarged brains, because species with longer reproductive periods have greater opportunities to reap the benefits of investment in learning during development (4), termed the “delayed benefits” hypothesis (3, 5), e.g. (6). Under the umbrella of the CBH, various elements of behavioural flexibility are emphasised as beneficial for survival in variable environments, including innovation (4), complex foraging strategies (7) and social learning (8). Common to all these ideas, however, is the prediction that across species, there should be a direct association between relatively enlarged brains and longer lifespans (5).

In addition to adaptive benefits, however, it is increasingly recognised that large brains impose costs, which may provide a sufficient explanation for the positive correlation between brain size and lifespan. The Developmental Costs Hypothesis (hereafter “DCH”) (9), together with the related ‘Maternal Energy’ hypothesis (10, 11) proposes that life history correlates of brain size reflect the need to extend development and maternal investment in order to build a large brain. Previous comparative analyses have provided support for this hypothesis: across mammals, pre-natal brain growth correlates specifically with gestation duration, while post-natal brain growth correlates specifically with lactation duration. Once these effects are accounted for, the brain size-lifespan correlation becomes non-significant, suggesting it is a side-effect of developmental costs (9). Positive correlations between adult brain size and pre- and post-natal developmental periods in birds are also consistent with the DCH (12). An additional ‘costs-based’ hypothesis, the ‘Expensive Brain’ framework, proposes a trade-off between the energetic costs of large brains and reproduction, such that large-brained species must spread higher costs of reproduction over longer lifespans (11). Common to both the Expensive Brain and Developmental Costs Hypotheses is the prediction that the brain size-lifespan correlation is indirect, mediated by relationships of both variables to protracted developmental periods.

To date, tests of these hypotheses have examined whole brain size. However, the brain is composed of functionally and anatomically heterogeneous structures which show heterochronicity in their developmental scheduling (13–16), influenced by structure-specific genes (17). The DCH therefore predicts that different brain structures should have specific developmental correlates across species. A comparative analysis in primates provides general support for this idea, finding that some brain components correlate more strongly with lifespan and some with age at first reproduction (1). However, this study did not examine specific developmental periods relevant to different aspects of brain growth, nor did it explicitly consider contrasting predictions made by costs-based and adaptive hypotheses. Furthermore, it did not control for phylogenetic non-independence in comparative data (1).

Here, we test the predictions of the DCH and CBH by examining life history correlates of brain size and the size of two major brain structures which together make up a substantial proportion of total brain size, and which have expanded relative to other structures during primate evolution: the neocortex and cerebellum (18). These two structures have to some extent evolved in a coordinated fashion, congruent with their anatomical

and functional connectivity (19) but also partly independently, in a mosaic fashion, with a notable acceleration in the rate of cerebellar expansion in the ape clade (20). These patterns of coordinated and mosaic brain component evolution are reflected in patterns of change at the molecular level, with similar numbers of changes in genes annotated during neocortical and cerebellar ontogeny in non-ape anthropoids, but more changes in cerebellar than neocortical genes during ape evolution (17). These phenotypic and genetic patterns imply that developmental mechanisms can be adjusted to facilitate a complex pattern of both coordinated and mosaic evolution of the cerebellum and neocortex.

Neurodevelopmental studies on humans and other primates suggest that one ontogenetic mechanism facilitating divergent evolution of these two structures is a substantial difference in the allocation of growth to pre- versus post-natal phases. While growth and neurogenesis of the neocortex is predominantly pre-natal, the cerebellum exhibits relatively rapid and prolonged post-natal neurogenesis and volumetric growth (21, 22). At birth, the human cerebellum is approximately 25% of its volume at two years of age, increasing by 240% in the first year post-natally, whilst the neonatal neocortex is already 46% of its volume at two years, increasing by a relatively modest 88% in the first year after birth (21). In terms of neurogenesis, the cerebellum is unusual in that the large majority (85%) of human cerebellar granule cells - the most numerous class of neurons in the brain - are generated post-natally (22). Post-natal growth of the human cerebellum is particularly extended relative to the cortex, attaining its peak volume at around 13.5 years (23). By contrast, the cortex reaches this milestone almost 5 years earlier at approximately 8.7 years (24).

Based on the literature discussed, we derive and test the following predictions. The CBH predicts that overall brain volume will be positively associated with lifespan, even when accounting for the effects of other life history phases. While previous tests of the CBH have focused on whole brain volume, some authors have suggested it may apply specifically to the neocortex, on the assumption that this region is particularly implicated in aspects of behavioural flexibility (5) such as innovation (25, 26), while the cerebellum not been predicted to play a major role. The DCH, in contrast, predicts that lifespan will not be positively associated with brain or brain component volumes after accounting for developmental effects. Further, owing to the differential allocation of developmental costs to pre- versus post-natal phases between the neocortex and cerebellum respectively, the DCH predicts that post-natal developmental periods (lactation duration and juvenile period) will be more strongly associated with cerebellum than with neocortex volume, and vice versa, pre-natal life history phases (gestation) will be more strongly associated with neocortex than with cerebellum volume. Given the markedly protracted post-natal development of the cerebellum, the DCH implies that the evolution of extended post-natal development in primates may be explained by the developmental costs associated with growing and maturing a large cerebellum in particular. Therefore the DCH also predicts that apes, a group with substantially expanded cerebella (20), will have significantly longer post-natal developmental periods than other primates, after accounting for allometric effects.

Methods

Brain volume data

We obtained whole brain, neocortex and cerebellum volumes (in mm³) for anthropoid species from an existing compilation (20), together with additional data for non-anthropoids compiled by the same authors (originally from (27, 28)), for 55 species. We analysed overall brain volume in addition to structure volumes to allow comparisons with previous work which has primarily investigated the whole brain (e.g. (1, 2, 8, 9)), and to give context to structure analyses in terms of how relationships between structure sizes and life history may be related to overall brain size. We did not use measures from a recent comparative dataset based on MRI scans (29) due to incompatible measures of neocortex volume (neocortical grey matter only in (29) versus whole neocortex volume in (20)) (see (30)).

Life history data

We obtained life history data from the PanTHERIA (31) and AnAge databases (32). For most life history traits – body mass (grams), gestation length (days), weaning age (days) and age at first birth (days) – we prioritised data from PanTHERIA, supplementing missing values with data from AnAge where possible. AnAge provides age of female sexual maturity rather than age of first birth estimates, but the two are very closely correlated among species with data for both variables (PGLS: $\beta=0.85$, $p<0.001$, $\lambda=0.00$, $n=43$). For lifespan data (estimated as maximum longevity), we prioritised records from AnAge due to higher data quality and longer estimates for many species compared with other datasets (33). Longevity records were converted from years (AnAge) or months (PanTHERIA) to days for comparability between datasets and with other life history traits. Some of these life history variables represent phases of life nested within one another (i.e. weaning age within age at first birth, age at first birth within lifespan). To avoid autocorrelation when including multiple life history predictors in the same model, we calculated two additional life history variables that do not overlap with any other for use in analyses: juvenile period and adult lifespan. We calculated juvenile period length by subtracting weaning age from age at first birth, and adult lifespan by subtracting age at first birth from maximum longevity.

After matching species across different datasets and to the 10ktrees primate phylogeny (34), the main sample contained 48 species (excluding humans) with complete data on all life history and brain volume variables. This dataset is available in the Electronic Supplementary Material (ESM data).

Statistical analyses

141 *Phylogenetic comparative methods*

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143 We tested predictions using comparative statistical methods that account for the influence of phylogeny,
144 specifically phylogenetic generalized least squares (PGLS) regression using functions from the caper R
145 package (35). We used a consensus phylogeny from 10ktrees (34). Pagel’s lambda (λ), a measure of
146 phylogenetic signal, was estimated by maximum likelihood. All continuous variables were log10 transformed
147 to reduce positive skew and improve fit to statistical assumptions. In all analyses, we treated brain or brain
148 structure volumes as the outcome variable and life history traits as the predictors. Additionally, in all analyses
149 we included body mass to control for allometric scaling of both brain structure volumes and life history traits
150 with body size (36–38). For analyses of structure volumes, we did not attempt to additionally control for
151 remaining brain volume for both theoretical and statistical reasons. The DCH rests on the assumption that
152 additional neural tissue, relative to body mass (reflective of energetic capacity), requires longer developmental
153 periods (9), and therefore does not make direct predictions about the size of brain components relative to one
154 another. We also focused on the size of brain components relative to body mass in order to facilitate direct
155 comparisons with prior tests of the DCH and CBH which have examined whole brain relative to body mass
156 (e.g. (2–4, 8, 9)). Further, measures of remaining brain volume are too highly correlated with body mass to
157 obtain confident estimates of the independent contributions of both remaining brain and body size to structure
158 volumes. Variance inflation factors (VIFs) were at least 15 when both body mass and remaining brain volume
159 were included as predictors of individual structures, exceeding commonly used thresholds for problematic
160 levels of collinearity (usually 5 or 10, (39)).

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162 We tested predictions by examining coefficients reported in global models and using model comparison to
163 identify more parsimonious models. For each brain volume measure, we first fit a global model including all
164 four life history traits plus body mass as predictor variables. Model performance was deemed acceptable for
165 all global models based on visual examination of diagnostic plots. VIFs for the global model ranged from 2.04
166 to 5.20, indicating moderate to potentially problematic levels of collinearity (although thresholds vary widely
167 in practice) (39). Then for each brain measure, we created a candidate set of models using functions from the
168 R package MuMIn (40). Candidate sets consisted of the full model, a null allometric (body mass only) model
169 and models containing all possible combinations of 1-3 life history predictors (total N=16 models for each
170 structure). Body mass was included in all candidate models to account for allometric relationships with brain
171 volumes and life history variables. Comparing PGLS models is complicated by the effect of phylogeny, since
172 both changes in the predictor variables and the influence of phylogeny can affect model fit. Therefore, to
173 simplify interpretation of life history effects, we fixed λ across all models in the candidate set for a given
174 structure, to the same value as that estimated by maximum likelihood from the global model (as recommended
175 in (35)). We used Bayesian Information Criterion (BIC) rather than AIC(c) scores to rank models, as the

former applies a higher penalty for additional parameters and is thus better suited to identifying the most parsimonious model (41). We report selected effect sizes to aid interpretation of the relative effects of life history phases, to illustrate comparisons that are particularly important in relation to predictions. We compare effects only between growth periods (i.e. gestation, lactation and juvenile period) as these comparisons are most biologically meaningful, and only when at least one of these variables has a significant or marginal effect ($p < 0.10$) in global models. Effect sizes for selected parameters were estimated by raising 10 to the power of their coefficients from the global models, which gives the amount of change in the dependent variable for a unit change a given predictor variable on the same scale as the data (e.g. number of additional mm³ in neocortex volume for an additional day of gestation), assuming the effects of all other predictors are held constant.

Differences between apes and other primates

We assessed the potential influence of apes (hominoidea) on the relationship between the cerebellum and life history variables in two different ways. First, we tested for differences in life history traits relative to body mass between apes and non-apes by fitting models predicting each life history trait in turn from body mass and a factor representing membership of the ape clade (following (20) which used this approach to examine such ‘grade shifts’ in cerebellar evolution). Here, we compared models in which either both slopes and intercepts, or intercepts only, are allowed to vary between apes and non-apes, using BIC scores. Second, we re-ran the global cerebellum model removing ape species ($n=5$) from the sample. To establish whether any differences in results were due specifically to removing apes versus reduced statistical power, we also re-ran the model 1000 times, removing 5 random non-ape species from the sample at each iteration. If the relationship between cerebellum volume and a particular life history variable is strongly contingent on the apes, we should expect that it generally remains significant when 5 random non-ape species are removed, but not when the 5 apes are removed.

Results

Figure 1 summarises the results of global models for all brain volume measures. Global models are reported in full in **Tables 1-3** while model selection tables are included in the Electronic Supplementary Material (**Tables S1-S3**).

Cognitive Buffer Hypothesis

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Contrary to the predictions of the Cognitive Buffer Hypothesis, we do not find a strong effect of adult lifespan on whole brain volume in the global model, in which the effects of pre- and post-natal development are taken into account (**Table 1**). In model comparisons, adult lifespan is retained in the best-supported model for overall brain volume, but the second-ranked model, containing gestation length only, is similarly well-supported (**Table S1**). We also find little support for the predictions of the CBH as applied to the neocortex. Gestation length is the strongest predictor of neocortex volume in the global model, while adult lifespan has little effect (**Table 2**). In model comparisons, adult lifespan is absent from the three highest-ranking models (**Table S2**). We do, however, find a significant effect of adult lifespan on cerebellum volume in the global model, after accounting for developmental periods (**Table 3**). In model comparisons, adult lifespan is included in the model with the lowest BIC score for cerebellum volume (**Table S3**), although the second-ranked, similarly supported model does not contain adult lifespan.

Developmental Costs Hypothesis

In the global model, gestation length is the strongest predictor of whole brain volume, while lactation and juvenile period have negligible effects (**Table 1**). In model comparisons, the two highest-ranked models retain gestation length, but not lactation duration or juvenile period (**Table S1**). Effect sizes from the model predict that brain volume increases by 3.18mm³ for each additional day of gestation, while by 1.17mm³ for each additional day of lactation, for a species with average life history traits and body mass. Gestation length is the only near-significant predictor of neocortex volume in the global model, while lactation and juvenile period have little to no effects (**Table 2**). Gestation length is the sole predictor included in the top-ranked model for neocortex volume, although the second highest ranking model contains both gestation length and weaning age (**Table S2**). Effect sizes predict a greater increase in neocortex volume for each additional day of gestation (4.11mm³) than lactation (1.30mm³), all else being equal. Conversely, cerebellum volume significantly increases with juvenile period in the global model, while gestation and lactation have negligible effects (**Table 3**). Juvenile period is retained in the 5 top-ranked models for cerebellum volume, and is the sole predictor in the second-highest ranked model (**Table S3**). Effect sizes from the global model suggest that for an average species, every additional day of life prior to first reproduction is associated with an increase of 1.85mm³ in cerebellum volume. We obtain estimates of zero phylogenetic signal in global models of cerebellum volume (**Table 3**). While a lack of phylogenetic signal is unexpected for evolutionarily conserved traits such as brain structure volumes, we show in supplementary analyses that this is unlikely to be the result of statistical artefacts (**ESM Appendix**).

Differences between apes and other primates

Relative to their body sizes, apes have significantly longer lactation and juvenile periods, and marginally longer adult lifespans, than non-apes (**Table 5, 6, 7; Figure 2, 3**). In contrast, apes do not significantly differ from non-apes in relative gestation time (**Table 4**). For all life history variables, BIC scores favoured intercept-only models over those in which both slopes and intercepts were allowed to vary between the clades (**Table 8**). When re-running the global cerebellum model without apes ($n=43$), the association between juvenile period and cerebellum volume becomes non-significant (**Table 9**). However, removing 5 randomly selected non-ape species from the sample also often results in a weakened relationship: p-values for juvenile period are 0.05 or greater in 64.7% of 1000 iterations. This suggests that the relationship between juvenile period and cerebellum volume is not solely contingent on the ape clade.

Discussion

Rather than a general extension of lifespan in large brained species, we find that specific aspects of life history are correlated with the volumes of different structures according to their developmental trajectories. Our results are therefore primarily consistent with predictions of the Developmental Costs and Expensive Brain hypotheses rather than the Cognitive Buffer Hypothesis, in that correlations between lifespan and brain size or neocortex volume appear to be by-products of the relationship of these structures with developmental periods. In support of the Developmental Costs Hypothesis more specifically, we find that brain structures with different emphases on pre-versus post-natal growth show predicted associations with those periods of investment. Maternal investment, specifically pre-natal investment, has an independent relationship with the relative volume of the neocortex. In contrast, cerebellum volume has an independent positive correlation with juvenile period length, congruent with the idea that interaction with the environment during maturation provides crucial input to the development of this structure, through play, for example. In summary, the correlation between brain size and life history in primates may require no specific adaptive explanation, instead reflecting the developmental mechanisms by which enlarged brains and brain components evolve.

Cognitive buffer hypothesis

We do not find evidence of a strong, direct correlation between brain size and lifespan, contradicting a central prediction of the CBH. Rather, the association between the two is weak when controlling for other life history phases. This finding is consistent with the interpretation that the brain size-lifespan correlation is confounded by the duration of maternal investment, as previously found across mammals (9) and more recently confirmed for primates in particular (42). We also find no evidence to support a direct association between lifespan and

neocortex volume, contradicting the idea that the neocortex plays a key role in extending lifespan via behavioural flexibility (5). We do find, however, an independent positive association of the cerebellum with adult lifespan, unanticipated by prior literature on the CBH, which has focused on whole brain or neocortex volume. Our findings could therefore be construed as consistent with a cognitive buffering effect specifically of the cerebellum. In support of this interpretation, the cerebellum is increasingly recognized to play a role not only in fine motor control and coordination but a wide diversity of cognitive functions including working memory, planning and decision-making (18). However, this finding is also consistent with the 'Neuronal Investment' hypothesis, which posits that longer-lived animals require larger brain volumes to compensate for longer periods of decline in neuronal function over their lifetimes (5). The cerebellum in particular may be implicated due its potentially greater susceptibility to neuronal loss with age compared with other structures (5). Further evidence, most crucially a relationship between cerebellum volume and survival, is thus required to distinguish between different explanations for this finding.

While we find little support for the CBH in terms of a direct link between overall brain size or neocortex size and lifespan in primates, our findings do not exclude the possibility that this hypothesis is supported by other lines of evidence, and in other taxonomic groups. For example, primate species with larger brains experience less variation in net energy intake than expected based on environmental seasonality compared with smaller-brained species, consistent with a cognitive buffering effect (43). Prior comparative work suggests that the extent to which brain size correlates with lifespan may vary between clades, finding greater support for the prediction among haplorrhine than strepsirrhine primates (1). A more recent comparative study finds support for the correlation within primates and rodents, but not other mammalian orders (3). Neither of these studies, however, accounted for the effects of developmental periods and therefore do not directly test the DCH. In birds, cognitive buffering effects are consistent with the findings that relatively large-brained species have lower adult mortality rates (44), experience more environmental variation (45) and have more stable populations in variable environments (46). Directly comparable tests of the DCH in primates and birds would therefore be a productive avenue of future research.

Developmental costs hypothesis

Our findings suggest that the relationship between lifespan and both overall brain volume and neocortical volume in primates is a by-product of maternal investment, primarily in pre-natal, rather than post-natal, offspring development. This result likely reflects the predominant role of pre-natal investment in neocortical growth specifically, given that this structure accounts for such a large proportion of overall brain volume. Consistent with this interpretation, and as predicted from the fact that neurogenesis and a relatively large

proportion of neocortical growth is completed before birth, the life history variable most strongly associated with adult neocortex size was gestation length. Our results suggest that evolutionary expansion of the neocortex is supported primarily by increased pre-natal investment, while cerebellar expansion requires greater investment in post-natal development. Despite the shared functional roles and correlated evolution of the cerebellum and neocortex (18–20, 47–49), a degree of independent, or mosaic, evolution of the two structures has been documented (20). Our results thus provide developmental mechanisms for the expansion of the neocortex and cerebellum, complementing evidence of distinct genetic mechanisms supporting such mosaic evolution (17). The patterns may be yet more complex, however: since the neocortex is composed of many heterogeneous systems, an interesting avenue for future work would be to investigate whether specific neocortical components or tissue types correlate with different aspects of maternal investment. Indeed, developmental scheduling varies across the neocortex, with occipital grey matter maturing earlier than that in the prefrontal cortex (50). Those specific areas and tissues which continue to grow post-natally may therefore be associated with post-natal developmental phases including lactation and juvenile period.

Adult cerebellum volume correlated positively with post-natal (juvenile) development, after accounting for variation in other life history phases. This pattern fits with evidence indicating late volumetric growth and maturation of this structure, extending through infancy and beyond in humans (23, 51, 52). At a cellular level, the post-natal genesis of the majority of cerebellar granule cells followed by synaptogenesis indicates high functional plasticity during this time, making environmental stimuli potentially critical in cerebellar maturation (22). Further evidence for the importance of environmental input in cerebellar development includes the low heritability of cerebellum volume compared to that of other brain structures (53), and effects of an impoverished post-natal environment on the volume of superior-posterior cerebellar lobes (54). Infancy and juvenility are periods of social learning, practice and play in an environment of reduced risk (55). Behaviourally, play is correlated with cerebellum volume (56) and with the volume of structures comprising the cortico-cerebellar system (57) across primates, and within species there are concurrent increases in the rate of play and formation of cerebellar synapses during post-natal development (58). The correlation between play and both post-natal brain growth and behavioural flexibility in primates is thus likely to involve cerebellar maturation (59, 60). Converging lines of evidence therefore suggest that many environmental influences on post-natal learning and development may be mediated by effects on the cerebellum.

Cerebellar expansion and extended maturation time in apes

When apes were removed from the PGLS analyses, the effect of juvenile period duration on cerebellum volume became non-significant. However, the same was true in the majority of cases when 5 random non-ape species were removed from the analyses, suggesting that this may be due to a loss of statistical power rather than contingency of results on the ape clade. We do, however, find that apes have a distinct life history profile compared to other primates, with significantly longer lactation and juvenile periods and marginally longer

adult lifespans. Together with prior evidence of accelerated cerebellar expansion in the apes (20), these results suggest that apes may have evolved extended post-natal maturation in part due to the need to invest in development of a large cerebellum and the time required for its experience-dependent maturation. The absence of a difference in relative gestation duration between the apes and other primates further suggests that ape life histories are distinct specifically in terms of their post-natal developmental trajectories. Consistent with this interpretation, cerebellar expansion in the apes is largely driven by enlargement of the cerebellar hemispheres: late-developing structures that are strongly implicated in the organisation and control of complex motor patterns (61, 62). Together, these results may help to explain the combination of unusually large cerebella (20) extended periods of immaturity (63) delayed locomotor independence (64), and high levels of social learning (65), play (66), extractive foraging and tool use (20) that characterises the ape clade. Future comparative analyses could investigate whether similar developmental profiles help explain independent cerebellar expansion in other mammalian lineages, such as elephants and cetaceans (67).

Conclusion

Developmental costs appear to provide the best explanation for the pattern of correlations between primate brain structures and life history. The central prediction of the Cognitive Buffer Hypothesis was not supported by strong, direct associations of lifespan and whole brain or neocortex volume; instead, these structures correlated most strongly with gestation length. The cerebellum does correlate with lifespan after accounting for developmental periods, consistent with an unanticipated cognitive buffering effect of this structure in particular, although alternative explanations are possible. Overall, we provide the first evidence that primate brain components exhibit distinct life history correlates that are congruent with their divergent developmental profiles. These divergent patterns support the view that selection on particular functional capacities can result in mosaic brain evolution mediated by complex developmental mechanisms (68).

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TABLES

Table 1: global model results for brain volume

Parameter	Estimate	S.E.	T-value	p-value
Intercept	0.28	0.72	0.39	0.70
Gestation	0.50	0.28	1.82	0.08
Lactation	0.07	0.07	0.97	0.34
Juvenile period	0.10	0.10	1.00	0.32
Adult lifespan	0.22	0.14	1.59	0.12
Body mass	0.51	0.05	10.10	<0.001

Table 1: Results of the global model for brain volume, predicted by all four life history traits and body mass ($N=48$, $R^2=0.88$, $\lambda=1$).

Table 2: global model results for neocortex volume

Parameter	Estimate	S.E.	T-value	p-value
Intercept	-0.19	0.83	-0.23	0.82
Gestation	0.61	0.32	1.93	0.06
Lactation	0.12	0.08	1.39	0.17
Juvenile period	0.08	0.11	0.76	0.45
Adult lifespan	0.18	0.16	1.13	0.27
Body mass	0.53	0.06	9.07	<0.001

Table 2: Results of the global model for neocortex volume, predicted by all four life history traits and body mass ($N=48$, $R^2=0.87$, $\lambda=1$).

Table 3: global model results for cerebellum volume

Parameter	Estimate	S.E.	T-value	p-value
Intercept	-0.94	0.65	-1.44	0.16
Gestation	0.08	0.22	0.39	0.70
Lactation	0.13	0.10	1.29	0.20
Juvenile period	0.27	0.13	2.01	0.05
Adult lifespan	0.32	0.16	2.06	0.05
Body mass	0.58	0.05	11.03	<0.001

Table 3: Results of the global model for cerebellum volume, predicted by all four life history traits and body mass ($N=48$, $R^2=0.96$, $\lambda=0$).

Table 4: comparison of gestation length in apes versus non-apes

Parameter	Estimate	S.E.	T-value	p-value
Intercept	1.93	0.08	25.55	<0.001
Body mass	0.09	0.02	4.26	<0.001
Ape vs. non-ape	0.08	0.07	1.19	0.24

Table 4: Results of the intercept-only model comparing gestation length relative to body mass in ape versus non-ape species ($N=48$, $R^2=0.34$, $\lambda=1$).

Table 5: comparison of lactation duration in apes versus non-apes

Parameter	Estimate	S.E.	T-value	p-value
Intercept	0.91	0.15	6.12	<0.001
Body mass	0.40	0.05	8.61	<0.001
Ape vs. non-ape	0.27	0.11	2.46	0.02

Table 5: Results of the intercept-only model comparing lactation duration relative to body mass in ape versus non-ape species ($N=48$, $R^2=0.75$, $\lambda=0.05$).

Table 6: comparison of juvenile period in apes versus non-apes

Parameter	Estimate	S.E.	T-value	p-value
Intercept	2.32	0.13	18.58	<0.001
Body mass	0.19	0.04	4.84	<0.001
Ape vs. non-ape	0.24	0.09	2.56	0.01

Table 6: Results of the intercept-only model comparing juvenile period length relative to body mass in ape versus non-ape species ($N=48$, $R^2=0.53$, $\lambda=0.26$).

Table 7: comparison of adult lifespan in apes versus non-apes

Parameter	Estimate	S.E.	T-value	p-value
Intercept	3.56	0.09	40.45	<0.001
Body mass	0.12	0.03	4.51	<0.001
Ape vs. non-ape	0.13	0.07	1.96	0.06

Table 7: Results of the intercept-only model comparing adult lifespan relative to body mass in ape versus non-ape species ($N=48$, $R^2=0.52$, $\lambda=0$).

Table 8: BIC scores for models comparing life history traits between apes and non-apes

Outcome variable	Intercept-only	Different slopes
Gestation	-121.79	-118.06
Lactation	-11.80	-10.21
Juvenile period	-37.60	-35.26
Adult lifespan	-59.57	-56.47

Table 8: BIC scores for models comparing gestation, lactation, juvenile period and adult lifespan relative to body size between apes and non-apes. In intercept-only models, only intercepts were allowed to vary between apes and non-apes, while in different slopes models, both intercepts and slopes were allowed to vary between apes and non-apes.

Table 9: global model results for cerebellum volume without ape species

Parameter	Estimate	S.E.	T-value	p-value
Intercept	-0.67	0.72	-0.92	0.36
Gestation	0.09	0.23	0.38	0.70
Lactation	0.09	0.11	0.79	0.44
Juvenile period	0.25	0.15	1.70	0.10
Adult lifespan	0.28	0.17	1.67	0.10
Body mass	0.59	0.06	10.19	<0.001

Table 9: results repeating the global model for cerebellum volume without ape species ($N=43$, $R^2=0.93$, $\lambda=0$).

FIGURE CAPTIONS

Figure 1: effects of life history predictors on brain structure volumes from global models

Figure 1: PGLS regression coefficients (points) with 95% confidence intervals (whiskers) for life history predictors of brain structure volumes, from global models. Dashed vertical lines indicate zero. Each row corresponds to a separate global model in which the brain structure volume on the Y axis is the outcome variable, predicted by the four life history traits on the X axis plus body mass (not shown).

Figure 2: relative lactation duration in apes versus non-apes

Figure 2: raw data (points) and regression slopes (lines) from a model predicting lactation duration from body mass, fitting separate intercepts for ape (green) and non-ape species (blue). Apes have significantly longer lactation periods relative to their body size than do non-apes.

Figure 3: relative juvenile period length in apes versus non-apes

Figure 3: raw data (points) and regression slopes (lines) from a model predicting juvenile period length from body mass, fitting separate intercepts for ape (green) and non-ape species (blue). Apes have significantly longer juvenile periods relative to their body size than do non-apes.

Brain volumes

Whole brain

Neocortex

Cerebellum

Gestation length

Weaning age

Juvenile period

Adult lifespan

Life history traits

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